

LETTERS TO THE EDITOR

- The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.
- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 600 words.
- In general, no letter should contain more than six references (also typed with double spacing).

Time of occurrence, duration, and ventricular rate of paroxysmal atrial fibrillation: the effect of digoxin

SIR,—I read with interest the excellent study of Rawles *et al* (1990;63:225–7) on the effect of digoxin in paroxysmal atrial fibrillation. It is always a pleasure to see widely held beliefs about a commonly used drug overturned by careful clinical observation. I would, however, take issue with one statement in their paper. Rawles *et al* point out that digoxin shortens the refractory period of atrial muscle, rendering the atrium more susceptible to fibrillation and increasing the rate of fibrillation. They suggest that the result of the use of digoxin in atrial fibrillation is “a compromise between increased fibrillation rate and reduced atrioventricular conduction, with the beneficial effect of reduced conduction generally predominating.”

In fact, the increase in atrial rate produced by digoxin may be beneficial in controlling ventricular response by increasing anterograde concealed conduction. The clinical existence of an increased atrial rate causing a decreased ventricular response is often noted when atrial flutter with 2:1 anterograde conduction converts to atrial fibrillation, when the increase in atrial rate results in a decreased ventricular rate.

Before conversion to sinus rhythm there is a coarsening in fibrillatory waves associated with a decrease in the number of wavelets in the atrium.¹ Experimentally this is associated with an increase in ventricular response²—an observation we have noted clinically.³ Indeed, not only may the effect of digoxin on atrial refractoriness be salutary for ventricular rate control, but some investigators have suggested that it is the predominant mechanism by which the drug slows the heart rate in atrial fibrillation.⁴

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- 1 Allesie MA, Rensome PL, Lammers WJEP, Kirchhoff CJHJ. The role of refractoriness, conduction velocity and wave length in initiation of atrial fibrillation in normal conscious dogs. In: Attuel P, Coumel P, Janse M, eds. *The atrium in health and disease*. Mt Kisco NY: Futura, 1989:27–42.
- 2 David D, Lang RM, Neumann A, Borow KM, Akselrod SA, Mor-Avi V. Parasympathetically modulated antiarrhythmic action of lidocaine in atrial fibrillation. *Am Heart J* 1990;119:1061–8.
- 3 Knowlton AA, Falk RH. Paradoxical increase in

heart rate before conversion to sinus rhythm in patients with recent onset atrial fibrillation. *Am J Cardiol* 1990;65:930–2.
4 Meijler FL. An ‘account’ of digitalis and atrial fibrillation. *J Am Coll Cardiol* 1985;5(suppl A):60A–8A.

The electrocardiogram does not reliably reflect the (anatomical) transmural extent of myocardial infarction

SIR,—Metcalf and colleagues (1990;63:267–72) presented an excellent six year follow up report of patients admitted to the coronary care unit with acute chest pain. The study was a well executed, retrospective investigation with useful results that accorded with our own recent observations. I wish to object to one small, but conceptually rather large, point. Metcalf *et al* repeat a chronic affliction of cardiologists—the continual muddling of anatomical subendocardial or non-transmural infarction and electrocardiographic non-Q wave infarction. It is well established that the electrocardiogram cannot reliably distinguish the anatomical mural extent of acute myocardial infarcts.^{1,2} Most Q wave infarcts are indeed anatomically transmural and some non-Q wave infarcts are anatomically non-transmural (mapping identifies additional Q wave zones in some “non-Q” infarcts in 12 lead recordings).

On page 270 Metcalf *et al* refer to “non-transmural infarction as we define it”, but nowhere does the text give an explicit definition; one must assume that they refer here, as elsewhere, to non-Q wave infarcts. In any case, confounding “non-transmural” or “subendocardial” with non-Q wave infarcts would be but a terminological quibble if it did not confuse concepts. The electrocardiogram is competent only to tell us if an infarct does or does not produce Q waves or other QRS changes.² We should therefore describe electrocardiographic results—the basis of Metcalf *et al*’s article—precisely as what is recorded, distinguishing clearly what we can see from what we may infer. The knowledgeable reader will know the range of inferences for any datum and the less well informed will be protected from unwarranted conclusions.

Finally, it was not surprising that Metcalf *et al* did not see “convergence of survival curves in the non-Q wave group” which had a lower mortality. They reasonably attribute this to the smaller infarcts associated with Q wave absence. Would they additionally comment on the convergence of late survival curves in the early reports distinguishing Q wave from non-Q wave infarction? Since that time has treatment advanced sufficiently to account for the divergent ultimate survival? Were Metcalf *et al*’s non-Q wave patients managed more vigorously with β blocking and calcium blocking agents or other treatments than would have been the case at the time when convergence of survival curves was reported?

These remarks are meant not so much as criticism but rather to amplify and clarify an excellent study.

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- 1 Phibbs B. “Transmural” vs. “subendocardial” myocardial infarction, an electrocardiographic myth. *J Am Coll Cardiol* 1983;1:561–4.
- 2 Spodick DH. Q-wave infarction versus ST-segment infarction: nonspecificity of ECG criteria for differentiating transmural and nontransmural lesions. *Am J Cardiol* 1983;51:913–5.

BOOK REVIEW

Pearls and Pitfalls in Electrocardiography. Pithy, Practical Pointers. Henry JL Marriott. (Pp 157; £12.62.) Philadelphia and London: Lea and Febiger, 1990. ISBN 0-8121-1334-9.

Those who roam the vast exhibition areas at the major American cardiologic meetings will certainly have encountered Dr Marriott at one of the booths, presenting material much like that contained in this book to a group in an open lecture theatre; they will have heard his light, yet earnest, admonitions about how much you can learn from careful scrutiny of electrocardiograms. Some of course may have read his larger books and already know of his original, sometimes idiosyncratic, yet always thoughtful, ideas. Until quite recently he would return to England every year or two and give a series of courses in London and the provinces. He makes a point of including nurses and technicians in the audience he aims to attract and always gives good value. The present book is no exception.

The first part of the title is drawn from an Oslerian phrase: “pearls are burrs that stick in the memory” and casts no aspersions on his readers. There are 70 sections, each covering one aspect, usually specific, occasionally general. Some of these segments are as brief as five lines but a few occupy more than one side of a page. Usually, but not invariably, the corresponding illustrations are opposite the text, so there is quite a lot of blank space. The individual items are important and the explanations usually, but not always, lucid. Occasionally a favourite phrase could have been less elegant but easier to understand, such as the heading for figure 39: “Anterograde conduction impaired by antecedent anterograde concealed conduction,” which takes a lot of working out. But most of the points are discussed in a helpful manner, and some of the pearls are true gems, like the very appropriate explanations of parasympathetic and atrioventricular dissociation. Occasionally Marriott uses the author’s privilege to trot out a hobby horse, an example being his personal definition of high grade block.

Many statements are supported by references and he sometimes highlights classic observations that deserve recall. Others, like the estimation of right ventricular pressure from V1, are unsupported. But his favourite source of wisdom is Sherlock Holmes, which gives you an idea of his approach. You will have to follow the master, too, when working out the numbering of the figures; the first is figure 2, a little disconcerting until you realise that each figure corresponds to a numbered item of text.

Worth while? Very definitely: an ideal Christmas present to the medical and nursing staff of a coronary care unit—excellent for systematic reading or for the occasional brief glance. And if you attend an American conference and hear that Barney Marriott is giving an extramural presentation, don’t miss it either. He is excellent on the rostrum, and this book gives you the flavour.

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